

1 **ROLE OF INTER-RELATED POPULATION-LEVEL HOST TRAITS IN**
2 **DETERMINING PATHOGEN RICHNESS AND ZONOTIC RISK**

3 TIM C.D. LUCAS^{1*†}, HILDE M. WILKINSON-HERBOTS² AND KATE E. JONES^{3*}

4 ¹Centre for Biodiversity and Environment Research, Department of Genetics, Evolution and Environ-
5 ment, University College London, Gower Street, London, WC1E 6BT, United Kingdom. 0000-0003-
6 4694-8107

7 ²Department of Statistical Science, University College London, Gower Street, London, WC1E 6BT,
8 United Kingdom.

9 ³Centre for Biodiversity and Environment Research, Department of Genetics, Evolution and Environ-
10 ment, University College London, Gower Street, London, WC1E 6BT, United Kingdom and Institute of
11 Zoology, Zoological Society of London, Regent's Park, London, NW1 4RY, United Kingdom. 0000-0001-
12 5231-3293

13 †Current address: Oxford Big Data Institute, Li Ka Shing Centre for Health Information and Discovery,
14 University of Oxford, Oxford, OX3 7FZ, United Kingdom.

15 *Corresponding authors: Tim C.D. Lucas: timcdlucas@gmail.com, Kate E. Jones: kate.e.jones@ucl.ac.uk

16 **Word count:** 6999

17 **Running header:** Host population-level drivers of pathogen richness

18

ABSTRACT

19 Zoonotic diseases are an increasingly important source of human infectious diseases, and host pathogen
20 richness of reservoir host species is a critical driver of spill-over risk. Population-level traits of hosts
21 such as population size, host density and geographic range size have all been shown to be important
22 determinants of host pathogen richness. However, empirically identifying the independent influences of
23 these traits has proven difficult as many of these traits directly depend on each other. Here we develop a
24 mechanistic, metapopulation, susceptible-infected-recovered model to identify the independent influences
25 of these population-level traits on the ability of a newly evolved pathogen to invade and persist in host
26 populations in the presence of an endemic pathogen. We use bats as a case study as they are highly
27 social and an important source of zoonotic disease. We show that larger populations and group sizes had
28 a greater influence on the chances of pathogen invasion and persistence than increased host density or
29 the number of groups. As anthropogenic change affects these traits to different extents, this increased
30 understanding of how traits independently determine pathogen richness will aid in predicting future
31 zoonotic spill-over risk.

32 **Keywords.** Pathogen competition, zoonotic disease, metapopulations, host pathogen richness, bats,
33 emerging infectious disease

34

1. INTRODUCTION

35 Zoonotic diseases are a major source of human infectious disease [1,2]. Epidemics of emerging, zoonotic
36 diseases pose a major threat to human health and economic development [3,4]. The probability of
37 zoonotic spill-over depends on, amongst other factors, the number of pathogen species carried by reservoir
38 host species (pathogen richness) [5]. Empirical, comparative studies across reservoir host species, suggest
39 that host morphological and life-history traits, such as large body size and longevity, correlate strongly
40 with high pathogen richness [6,7]. However, traits related to reservoir host population biology are also
41 expected to affect disease dynamics and therefore influence pathogen richness. Population-level traits
42 such as increased host density [6,8,9], large geographic range size [6,9,10] and greater population structure
43 (nonrandom interactions between individuals) [10,11] have been shown to correlate with high pathogen
44 richness, although the evidence for a relationship with group size (number of individuals in a social
45 group) has been equivocal in many studies [9,10,12–14]. Population size (total number of individuals), an
46 important population-level trait, has rarely been included in comparative studies, despite its importance
47 in describing epidemiological populations [15].

48 Collinearity between explanatory variables is a common problem in correlative studies, and this issue
49 is exacerbated when there are clear, causal relationships between explanatory variables. There are two
50 particularly clear relationships between the population-level traits associated with pathogen richness.
51 Firstly, host density, d , host population size, N , and geographic range size, a , are, by definition, linked
52 by $d = N/a$ (see electronic supplementary material, table S1 for all parameters used) and this relationship
53 has broad empirical support [16]. Secondly, host population size can be decomposed into two components,
54 the number of groups, m , and the average size of a group, n , with $N = mn$. Correlative, comparative

55 studies would be especially poor at identifying which, if any, of these traits causally affect pathogen
56 richness. This lack of discriminatory power is particularly important with respect to global change and
57 its effects on zoonotic disease emergence. Population-level traits such as population size and geographic
58 range size, although interrelated, will respond differently to global change and the response will be species
59 specific. Some host species may suffer geographic range contractions while their density remains constant
60 [17]. Other host species might retain their geographic range but have a depressed population density
61 [18]. Only by knowing which of these interrelated traits control pathogen richness will we be able to
62 predict future changes in pathogen richness.

63 Mechanistic models provide one method for comparing the importance of intrinsically related traits
64 and can provide a deeper understanding of the system than correlative approaches. Theoretical studies
65 have established that a number of host population-level traits are important for epidemiological dynamics
66 and the maintenance of pathogen richness. In particular, host density, population structure and group
67 size are well established as having central roles in pathogen dynamics [19–21]. A number of studies have
68 found that increased host population structure can promote pathogen coexistence [22–24]. While these
69 studies have examined whether these population-level traits can promote pathogen richness, none have
70 attempted to distinguish which might be the most important. Mechanistic models that try to disentangle
71 the interplay between population-level traits including host density, population size, geographic range
72 size, group size and the number of groups are critically needed.

73 Here, we use multipathogen models to individually vary host population-level traits. We examine a
74 simple deterministic model to establish whether a newly evolved pathogen can invade into an unstructured
75 population in the presence of strong competition from an endemic pathogen strain. We then examine a
76 stochastic, metapopulation model that was parameterised to broadly mimic wild bat populations. We
77 used bats as a case study as group (colony) size is very variable between bat species and bat colonies
78 are often very stable [25–28]. Furthermore, bats are particularly relevant in the context of zoonotic
79 disease as they are thought to be reservoirs for a number of recent, important outbreaks [29,30]. We
80 examined how the interrelated population-level traits affect the ability of a newly evolved pathogen
81 to invade and persist in a population. We used these simulations to examine two specific hypotheses.
82 First, we investigated whether host population size or density more strongly promotes the invasion of
83 a new pathogen. Secondly, we investigated whether the invasion of a new pathogen is more strongly
84 promoted by group size or the number of groups. We found that population size has a much stronger
85 effect on the invasion of a new pathogen than host density. We also found that increasing population
86 size by increasing group size promotes pathogen invasion much more than increasing population size by
87 increasing the number of groups.

88

2. METHODS

89 **(a). Two pathogen SIR model.** We developed a multipathogen, susceptible-infected-recovered (SIR)
90 compartment model to examine the probability that a newly evolved pathogen would invade and persist
91 into a population in the presence of an identical, endemic pathogen. Susceptible individuals were counted
92 in class S (figure 1) while infected individuals were counted in classes I_1 , I_2 and I_{12} , being individuals

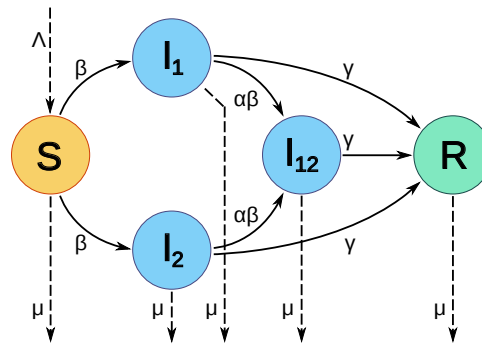


FIGURE 1. Schematic of the two-pathogen SIR model used. Individuals are in one of five epidemiological classes, susceptible (orange, S), infected with Pathogen 1, Pathogen 2 or both (blue, I_1 , I_2 , I_{12} , respectively) or recovered and immune from further infection (green, R). Transitions between classes occur as indicated by solid arrows and depend on transmission rate (β), coinfection adjustment factor (α) and recovery rate (γ). Births (Λ) and deaths (μ) are indicated by dashed arrows. Note that individuals in I_{12} move into R , not back to I_1 or I_2 . That is, recovery from one pathogen causes immediate recovery from the other pathogen.

93 infected with Pathogen 1, Pathogen 2 or both, respectively. Recovered individuals, R , were immune to
94 both pathogens, even if they had only been infected by one (i.e. there was complete cross-immunity).
95 Furthermore, recovery from one pathogen moved an individual into the recovered class, even if the
96 individual was coinfecting (figure 1). Though our study was restricted to two pathogens, this modelling
97 choice allows the model to be easily expanded to include many pathogens. Our assumption of immediate
98 recovery from all other pathogens is likely to be reasonable [31] as any up-regulation of innate immune
99 response or acquired immunity would affect both pathogens equally. The coinfection rate (the rate at
100 which an infected individual is infected with a second pathogen) was adjusted compared to the infection
101 rate by a factor α . Birth rate (Λ) was set equal to the death rate (μ), meaning the population did not
102 systematically increase or decrease. New born individuals entered the susceptible class. Infection and
103 coinfection were assumed not to cause increased mortality as bats show no clinical signs of infection for
104 a number of viruses [32,33].

105 Population structure is present in bat populations as demonstrated by the existence of subspecies, mea-
106 surements of genetic dissimilarity and behavioural studies [25,26,34]. Therefore assuming a fully-mixed
107 population is an oversimplification. Consequently, the population was modelled as a metapopulation
108 network with groups being nodes and dispersal between groups being indicated by edges (electronic sup-
109plementary material, figure S1). Individuals within a group interacted randomly so that the group was

110 fully mixed. Dispersal occurred at a rate ξ between groups connected by an edge in the network. The
111 dispersal rate from a group y with degree k_y to group x was ξ/k_y . Note that this rate was not affected
112 by the degree and size of group x and the total rate of dispersal was not affected by the degree of a
113 group. We examined the full model using stochastic, continuous-time simulations, in R [35,36]. The full
114 details of the model are given in electronic supplementary material S1.

115 **(b). Deterministic model.** We examined a single-group, deterministic model to establish a baseline
116 expectation for whether a newly evolved pathogen strain could invade into a population in the presence
117 of an identical strain given the assumptions of our two-pathogen SIR model (for details see electronic
118 supplementary material S2). If we first consider the endemic pathogen (Pathogen 1) we have a typical
119 SIR model with vital dynamics [37] with equilibrium values $S^* = \frac{\mu+\gamma}{\beta}$ and $I_1^* = \frac{\Lambda n}{\gamma+\mu} - \frac{\mu}{\beta}$ where β and
120 γ are the transmission and recovery rates and n is the group size. When Pathogen 2 is introduced, its
121 rate of change can be written as

$$\frac{dI_2}{dt} = \beta S^* I_2 + \alpha \beta I_1^* I_2 - (\gamma + \mu) I_2 \quad (1)$$

122 which is greater than zero when $\alpha (\Lambda R_0 - \mu) I_2 > 0$ (with $R_0 = \frac{\beta N}{\gamma+\mu}$ being the basic reproduction number
123 and being equal for the two identical pathogens). As $\Lambda = \mu$ due to the assumption of a stable population
124 size, $\Lambda R_0 - \mu$ is greater than zero, Therefore, $\frac{dI_2}{dt}$ is greater than zero provided α is greater than zero.
125 That is, provided cross-immunity is not complete, Pathogen 2 will invade in this deterministic model.
126 This means that it is only stochastic extinction that would prevent a pathogen from invading into a
127 population. Our more detailed simulations are therefore examining how effectively different population-
128 level traits alleviate stochastic extinction or allow longer term persistence.

129 **(c). Parameter selection.** While some fixed parameters were chosen to approximate those found in
130 wild bat populations, others were chosen for modelling reasons. Assuming equal birth and death rates, Λ
131 and μ , were both set to 0.05 per year giving a generation time of 20 years [27,28]. Setting birth and death
132 rates equal gives a stochastically varying population size but given the length of the simulations groups
133 were very unlikely to go extinct. Although it is difficult to directly estimate infection durations in wild
134 bat populations [38], evidence suggests these can be on the scale of days [39] up to months or years [40–
135 42]. Here we set the infection duration, $\frac{1}{\gamma}$, to one year which is a long lasting, but non-chronic, infection.
136 As estimating transmission rates is particularly difficult we used three values of the transmission rate, β :
137 0.1, 0.2 and 0.3. These values were chosen as very high values of R_0 were required so that a reasonable
138 number of simulations experienced an invasion of Pathogen 2. The coinfection adjustment parameter,
139 α , was set to 0.1. The deterministic model showed that $\alpha = 0$ and $\alpha > 0$ are qualitatively different
140 with the number of individuals infected with Pathogen 2 being stable and increasing respectively. The
141 case where Pathogen 2 does not invade and spread ($\alpha = 0$) is not important for pathogen richness so we
142 chose a small, non-zero value for α . The dispersal rate, ξ , was set to 0.01 which yields 17% of individuals
143 dispersing in their lifetime. This relates to a species with juvenile dispersal of a proportion of males and
144 very few females [28,43].

145 The effect of geographic range size on disease dynamics occurred through changes in the metapopula-
146 tion network. Dispersal was only allowed to occur between two groups if they were connected nodes in
147 the metapopulation network. The metapopulation network was created for each simulation by randomly
148 placing groups in a square space which represented the geographic range of the species (electronic sup-
149plementary material, figure S1). Groups within a threshold distance of each other were connected in the
150 metapopulation network. This meant that the metapopulation network was not necessarily connected
151 (made up of a single connected component). To ensure connected metapopulation networks would have
152 required repeatedly resampling the group locations until a connected metapopulation occurred. How-
153 ever, this would have biased the mean degree, \bar{k} . Therefore, it was considered preferential to keep the
154 unconnected networks.

155 **(d). Experimental setup.** A total of 4500 simulations were run. In each simulation, each group in
156 the host population was seeded with 20 individuals infected with Pathogen 1. A ‘burn-in’ of 6×10^5
157 events was run to allow Pathogen 1 to spread and reach equilibrium. Plotting of preliminary simulations
158 was used to determine that 6×10^5 events was enough to ensure equilibrium. After this burn-in, five
159 host individuals infected with Pathogen 2 were added to one randomly selected group. The invasion and
160 persistence of Pathogen 2 was considered successful if any individuals infected with Pathogen 2 remained
161 at the end of the simulation. As simulations with many individuals and infection events had more events
162 per unit time, the end of the simulation had to be defined in terms of time rather than the number
163 of events. Simulations were run until 75 years had elapsed since the introduction of Pathogen 2. This
164 value was chosen so that pathogens had to persist for multiple host generations in order to be considered
165 persistent.

166 Three sets of 1500 simulations were run in which three population parameters were varied: (*i*) ge-
167 ographic range size (with corresponding change in host density), (*ii*) group size (with corresponding
168 change in population size), and (*iii*) the number of groups (with corresponding change in population
169 size). To allow comparisons between simulation sets, the parameter that was being varied in each set
170 was assigned its default value multiplied by 0.25, 0.5, 1, 2 and 4. To examine whether host density had
171 a stronger effect on pathogen invasion than population size, results from simulation set *i* were compared
172 to those from sets *ii* and *iii*. To examine whether group size or the number of groups was the more
173 important component of population size, results from simulation set *ii* were compared to those from *iii*.

174 The spatial scale is arbitrary; only the ratio between the geographic range size and the threshold dis-
175 tance for groups being connected in the metapopulation network had any effect on simulation outcomes.
176 We parameterised the spatial scale by arbitrarily selecting a threshold distance of 100 km. . The default
177 (10000 km^2) and upper and lower bounds of the geographic range size ($2500\text{--}40000 \text{ km}^2$) were then se-
178 lected to maximise the range of \bar{k} (electronic supplementary material, figure S2) while not having many
179 simulations with networks that were unconnected. That is, we aimed for low host density populations
180 to have relatively sparse population networks, while high host density populations had fully-connected
181 metapopulation networks. This reflects the existence of both isolation-by-distance [43–45] and panmixia
182 [29,46,47] in bat species. The default group size was 400 with a range of 100–1600 which is representative
183 of many bat species [27]. The default number of groups was 20 with a range of 5–80. This minimum

184 value is close to the minimum possible for the population to still be considered a metapopulation, while
185 the maximum value was constrained by computational costs.

186 In the first set of simulations (*i*), host density was varied by keeping population size constant ($N =$
187 8000, $n = 400$, $m = 20$) while varying geographic range size. The values of geographic range size used
188 were 2500, 5000, 10000, 20000 and 40000 km² which gave density values of 3.2, 1.6, 0.8, 0.4 and 0.2
189 animals per km². In the second set of simulations (*ii*), population size was varied by changing group size
190 while the number of groups was kept constant. To keep host density constant, geographic range size was
191 increased as population size increased. The values of group size used were 100, 200, 400, 800 and 1600
192 while geographic range size was set to 2500, 5000, 10000, 20000 and 40000 km². This gave population
193 sizes of 2000, 4000, 8000, 16000 and 32000 while host density remained at 0.8 hosts per km². In the
194 third set of simulations (*iii*), population size was varied by changing the number of groups while group
195 size was kept constant. Again, to keep host density constant, geographic range size was increased as
196 population size increased. The numbers of groups used were 5, 10, 20, 40 and 80 while geographic range
197 size was set to 2500, 5000, 10000, 20000 and 40000 km². Again, this gave population sizes of 2000, 4000,
198 8000, 16000 and 32000 while host density remained at 0.8 hosts per km².

199 **(e). Statistical analysis.** For each set of simulations, we fitted binomial GLMs in R [35] with the
200 proportion of invasions of Pathogen 2 as the response variable. To enable comparison between GLMs we
201 divided the explanatory variables by their default values and log₂ transformed. The explanatory variables
202 for all three sets of simulations therefore became evenly spaced between -2 and 2. To investigate whether
203 an increase in host population size created a stronger increase in probability of pathogen invasion than
204 an equal increase in host density we compared the size (and 95% confidence intervals) of the regression
205 coefficients of host density (*i*) to group size (*ii*) and number of groups (*iii*). To examine whether an
206 increase in group size creates a stronger increase in invasion probability than a proportionally equal
207 increase in number of groups we compared regression coefficients of group size (*ii*) to number of groups
208 (*iii*).

209 3. RESULTS

210 **(a). Population size and host density.** The estimated GLM coefficients were always larger for sim-
211 ulations where population size was varied (sets *ii* and *iii*) than when host density (set *i*) was varied
212 (table 1, electronic supplementary material figure S3). Increasing population size, either by increasing
213 group size or number of groups, increased the probability of invasion (figure 2). The positive relationship
214 between group size and invasion was strong and significant at all transmission rates, while the rela-
215 tionship between number of groups and invasion was weaker but still significant. In contrast, varying
216 host density did not significantly alter invasion probability except for when $\beta = 0.3$ where there was a
217 significant decrease in invasion probability with increased host density (GLM: coefficient = -0.33, $p =$
218 0.04). The 95% confidence intervals for the coefficients of group size did not overlap with those for the
219 coefficients of host density at any value of β while the 95% confidence intervals for coefficients of number
220 of groups only overlapped with those for host density at $\beta = 0.2$.

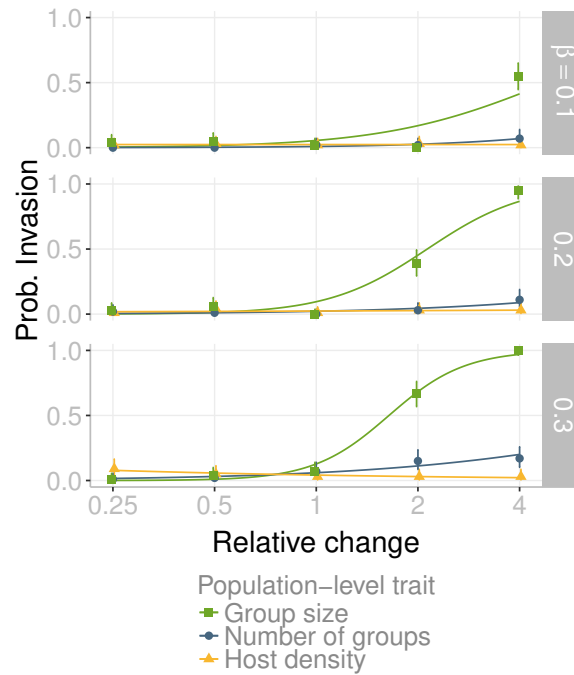


FIGURE 2. Comparison of the effect of population-level traits on probability of invasion. Population-level traits are group size (green lines, squares), number of groups (blue lines, circles) and host density (yellow lines, triangles). The x -axis shows the change ($\times 0.25$, 0.5, 1, 2 and 4) in each of these traits relative to the default value. Default values are: number of groups = 20, group size = 400 and host density = 0.8 animals per km^2 . Each point is the mean of 100 simulations and bars are 95% confidence intervals. Each curve was obtained by fitting a binomial GLM. Relationships are shown separately for each transmission value, β .

221 **(b). Group size and number of groups.** In all cases, the estimated GLM coefficients were larger
222 for simulations where group size was varied (set *ii*) than when the number of groups (set *iii*) was varied
223 (table 1, electronic supplementary material figures S3 and S4). Increasing either group size or the number
224 of groups increased the probability of invasion but this effect was stronger for group size (figure 2). The
225 95% confidence intervals of regression coefficients for group size did not overlap with those for number of
226 groups for the simulations with $\beta = 0.2$ or 0.3 but the confidence intervals did overlap for the simulations
227 when $\beta = 0.1$.

TABLE 1. Regression results comparing effects of group size, number of groups and host density. Estimated regression coefficients and their 95% confidence intervals are from binomial GLM regressions with the proportion of invasions as the response variable and all explanatory variables being standardised by dividing by the default parameter value and applying a \log_2 transform. Group size and number of groups were varied while keeping density equal while density was varied by changing geographic range size while keeping population size equal. Results are given for three transmission (β) values.

β	Variable	Coefficient	(95% CI)	p
0.1	Group size	1.24	(0.94, 1.59)	$< 10^{-4}$
	Number of groups	1.03	(0.43, 1.87)	3.71×10^{-3}
	Density	0.00	(-0.41, 0.41)	1.00
0.2	Group size	2.06	(1.71, 2.47)	$< 10^{-4}$
	Number of groups	0.73	(0.32, 1.24)	1.54×10^{-3}
	Density	0.13	(-0.28, 0.55)	0.54
0.3	Group size	2.69	(2.23, 3.22)	$< 10^{-4}$
	Number of groups	0.68	(0.42, 0.98)	$< 10^{-4}$
	Density	-0.33	(-0.66, -0.03)	0.04

228

4. DISCUSSION

229 Overall, our results suggest that population size strongly promotes pathogen richness. In contrast, host
 230 density was found to weakly promote pathogen richness if at all. This is in agreement with theoretical
 231 arguments that population size is the more natural description of populations [15] but is in contrast to
 232 the many comparative studies that find host density to be a significant predictor of pathogen richness
 233 [6,8,9]. This suggests that in these comparative studies, host density is acting as a proxy for population
 234 size rather than being a causal factor. Alternatively, the local measurements of host density could be
 235 indicative of some other trait such as maximum host density, rather than global density. Our results also
 236 suggest that a population made up of large groups would have higher pathogen richness than a population
 237 made up of many smaller groups. Comparative studies examining group size have had conflicting results
 238 [9,10,12,13] with a meta-analysis finding a non-significant relationship between group size and pathogen
 239 richness [14]. The largest study specifically on bats found a negative relationship between group size and
 240 pathogen richness [13]. Therefore while our results regarding group size are potentially in conflict with
 241 the empirical literature, more definitive empirical studies are needed. This conflict may also indicate a
 242 difference between pathogen richness accumulation (the focus of this study) and total pathogen richness.

243 Our results also suggest that host geographic range size does not promote pathogen richness, yet a
 244 number of studies have found evidence of this relationship [6,9]. In studies that do not also include
 245 population size or host density, geographic range size may be acting as a proxy for population size.

246 Alternatively this empirical association may be because in wild species, increased host geographic size
247 tends to entail a greater variety of environmental conditions and a greater number of sympatric species
248 which is known to also affect pathogen richness [48] and these factors are not considered in this study.
249 Finally, we found an unexpected negative association between density and invasion probability at $\beta = 0.3$.
250 Due to the small size of the estimated coefficient, the marginal p-value and the lack of a consistent
251 relationship at other β values, we suspect that this is not a real effect but merely due to chance. However,
252 given that in our study, increased density affects disease dynamics by decreasing population structure, this
253 negative relationship does fit with studies that suggest that population structure should allow pathogen
254 coexistence [10,11,23].

255 Many comparative studies measure population-level traits, yet it is rarely considered how these might
256 be causally related (though statistical correlations between explanatory variables are often handled ap-
257 propriately). For example, host density is often used in studies [8,9,49], yet host density is directly
258 associated with population size. Our results suggest that despite the association between these two
259 traits, they are not equivalent. These causal relationships between population-level traits should be
260 considered more carefully in future comparative studies. Researchers could include of an interaction
261 term between geographic range size and host density to test for the importance of population size, for
262 example.

263 This study was limited to one mechanism by which pathogen richness can be increased; the invasion
264 and persistence of a newly evolved pathogen [50]. However, other processes such as pathogen extinction
265 are also likely to be important [50]. We also restricted ourselves to the context of competition between
266 two pathogens in a social host species. As the model was of a directly transmitted pathogen there is
267 no transmission between individuals in separate groups. Infection via shared food sources or contact
268 between individuals from different groups (e.g., during swarming [28]) would act to reduce population
269 structure and therefore host density might become more important. However, further modelling would
270 be required to demonstrate this while only empirical studies would be able to indicate the true relative
271 importance of these different transmission routes in wild populations.

272 It is clear that many species are suffering strong population changes due to global change [17]. These
273 changes might affect geographic range size [17], population size [18], population connectivity [51,52] or
274 group size [53,54] to different degrees. The monitoring of these different aspects of population change,
275 especially in bats, can often be difficult and may require further developments in monitoring to be
276 effective, for example developing methods that use data from acoustic detectors [55–57]. Our results
277 suggest that pathogen communities will respond differently depending on which traits are most strongly
278 affected by global change. In short, species suffering reductions in groups size [53,54] are predicted to
279 experience a decrease in pathogen richness in the long term and there is some evidence that this process
280 is occurring [10,58]. Species that are experiencing an increase in group size [53] would be expected to
281 gain new pathogen species and therefore pose a greater risk of being the source of a zoonotic disease. In
282 contrast, species suffering a reduction in geographic range size [17] or a decrease in population size [18],
283 without a corresponding decrease in group size, are expected to experience smaller changes in pathogen

284 richness. Only by examining the mechanisms that control pathogen richness can we understand and
285 predict these changes.

286 DATA ACCESSIBILITY

287 The implementation of the model is available as an R package on GitHub [36]. This can be found at
288 <https://github.com/timcdlucas/MetapopEpi>. All other code and simulation output data is available on
289 GitHub at <https://github.com/timcdlucas/Abundance-Density-Manuscript>.

290 COMPETING INTERESTS

291 We have no competing interests.

292 AUTHOR'S CONTRIBUTIONS

293 TCDL wrote the simulations and performed the analysis. TCDL, HMH and KEJ co-designed the
294 study. TCDL drafted the manuscript. TCDL, HMH and KEJ all edited the manuscript and gave final
295 approval for publication.

296 ACKNOWLEDGEMENTS

297 We thank Andy Fenton and David Murrell for comments on the manuscript and help with the an-
298 alytical model. This work, The Dynamic Drivers of Disease in Africa Consortium, NERC project no.
299 NE-J001570-1 was funded with support from the Ecosystem Services for Poverty Alleviation Programme
300 (ESPA). The ESPA programme is funded by the Department for International Development (DFID), the
301 Economic and Social Research Council (ESRC) and the Natural Environment Research Council (NERC).

302 FUNDING

303 This study was funded through a CoMPLEX PhD studentship at University College London supported
304 by BBSRC and EPSRC (TCDL). KEJ was funded by the Ecosystem Services for Poverty Alleviation
305 Programme (ESPA) (NE-J001570-1).

306 REFERENCES

- 307 [1] Jones KE, Patel NG, Levy MA, Storeygard A, Balk D, Gittleman JL, and Daszak P 2008. Global
308 trends in emerging infectious diseases. *Nature* **451**, 990–993. (10.3201/eid0903.020336).
- 309 [2] Taylor LH, Latham SM, and Mark E 2001. Risk factors for human disease emergence. *Phil. Trans.*
310 *R. Soc. B* **356**, 983–989. (10.1093/acprof:oso/9780198525738.003.0035).
- 311 [3] World Health Organization et al. 2016. WHO: Ebola situation report. ([http://apps.who.int/ebola/current-
312 situation/ebola-situation-report-30-march-2016](http://apps.who.int/ebola/current-situation/ebola-situation-report-30-march-2016)).
- 313 [4] World Bank 2015. World Development Indicators: Size of the economy. (<http://wdi.worldbank.org/table/1.1>).
- 314 [5] Wolfe ND, Eitel MN, Gockowski J, Muchaal PK, Nolte C, Tassy Prosser A, Ndongo Torimiro J,
315 Weise SF, and Burke DS 2000. Deforestation, hunting and the ecology of microbial emergence. *Global
316 Change & Human Health* **1**, 10–25. (10.1023/A:1011519513354).

-
- 317 [6] Kamiya T, O'Dwyer K, Nakagawa S, and Poulin R 2014. What determines species richness of
318 parasitic organisms? A meta-analysis across animal, plant and fungal hosts. *Biol. Rev.* **89**, 123–134.
319 (10.1111/brv.12046).
- 320 [7] Luis AD, Hayman DTS, O'Shea TJ, Cryan PM, Gilbert AT, Pulliam JRC, Mills JN, Timonin ME,
321 Willis CKR, Cunningham AA, et al. 2013. A comparison of bats and rodents as reservoirs of zoonotic
322 viruses: are bats special? *Proc. R. Soc. B* **280**, 20122753. (10.1098/rspb.2012.2753).
- 323 [8] Morand S and Poulin R 1998. Density, body mass and parasite species richness of terrestrial mam-
324 mals. *Evol. Ecol.* **12**, 717–727. (10.1023/A:1006537600093).
- 325 [9] Nunn CL, Altizer S, Jones KE, and Sechrest W 2003. Comparative tests of parasite species richness
326 in primates. *Am. Nat.* **162**, 597–614. (10.1086/378721).
- 327 [10] Turmelle AS and Olival KJ 2009. Correlates of viral richness in bats (order Chiroptera). *EcoHealth*
328 **6**, 522–539. (10.1007/s10393-009-0263-8).
- 329 [11] Maganga GD, Bourgarel M, Vallo P, Dallo TD, Ngoagouni C, Drexler JF, Drosten C, Nakouné
330 ER, Leroy EM, and Morand S 2014. Bat distribution size or shape as determinant of viral richness
331 in african bats. *PLoS One* **9**, e100172. (10.1371/journal.pone.0100172).
- 332 [12] Vitone ND, Altizer S, and Nunn CL 2004. Body size, diet and sociality influence the species richness
333 of parasitic worms in anthropoid primates. *Evol. Ecol. Res.* **6**, 183–199.
- 334 [13] Gay N, Olival KJ, Bumrungsri S, Siriaroonrat B, Bourgarel M, and Morand S 2014. Parasite and
335 viral species richness of Southeast Asian bats: fragmentation of area distribution matters. *Int. J.*
336 *Parasitol. Parasites Wildl.* **3**, 161–170. (10.1016/j.ijppaw.2014.06.003).
- 337 [14] Rifkin JL, Nunn CL, and Garamszegi LZ 2012. Do animals living in larger groups experience
338 greater parasitism? A meta-analysis. *Am. Nat.* **180**, 70–82. (10.1086/666081).
- 339 [15] Begon M, Bennett M, Bowers RG, French NP, Hazel S, and Turner J 2002. A clarification of
340 transmission terms in host-microparasite models: numbers, densities and areas. *Epidemiol. Infect.*
341 **129**, 147–153. (10.1017/S0950268802007148).
- 342 [16] Blackburn TM, Cassey P, and Gaston KJ 2006. Variations on a theme: sources of heterogeneity
343 in the form of the interspecific relationship between abundance and distribution. *J. Anim. Ecol.*
344 **75**, 1426–1439. (10.1111/j.1365-2656.2006.01167.x).
- 345 [17] Thomas CD, Cameron A, Green RE, Bakkenes M, Beaumont LJ, Collingham YC, Erasmus BF,
346 De Siqueira MF, Grainger A, Hannah L, et al. 2004. Extinction risk from climate change. *Nature*
347 **427**, 145–148. (10.1038/nature02121).
- 348 [18] Craigie ID, Baillie JE, Balmford A, Carbone C, Collen B, Green RE, and Hutton JM 2010. Large
349 mammal population declines in Africa's protected areas. *Biol. Cons.* **143**, 2221–2228. (10.1016/j.biocon.2010.06.007).
- 350 [19] Colizza V and Vespignani A 2007. Invasion threshold in heterogeneous metapopulation networks.
351 *Phys. Rev. Lett.* **99**, 148701. (10.1103/physrevlett.99.148701).
- 352 [20] May RM and Anderson RM 1979. Population biology of infectious diseases: Part II. *Nature*
353 **280**, 455–461. (10.1038/280455a0).
- 354 [21] Anderson RM and May RM 1979. Population biology of infectious diseases: Part I. *Nature* **280**, 361–
355 7. (10.1038/280361a0).

-
- 356 [22] Qiu Z, Kong Q, Li X, and Martcheva M 2013. The vector–host epidemic model with multiple
357 strains in a patchy environment. *J. Math. Anal. Appl.* **405**, 12–36. (10.1016/j.jmaa.2013.03.042).
- 358 [23] Allen LJ, Kirupaharan N, and Wilson SM 2004. SIS epidemic models with multiple pathogen
359 strains. *J. Differ. Equ. Appl.* **10**, 53–75. (10.1080/10236190310001603680).
- 360 [24] Nunes A, da Gama MT, and Gomes M 2006. Localized contacts between hosts reduce pathogen
361 diversity. *J. Theor. Biol.* **241**, 477–487. (10.1016/j.jtbi.2005.12.010).
- 362 [25] Kerth G, Perony N, and Schweitzer F 2011. Bats are able to maintain long-term social relationships
363 despite the high fission–fusion dynamics of their groups. *Proc. R. Soc. B* **278**, 2761–2767.
- 364 [26] McCracken GF and Bradbury JW 1981. Social organization and kinship in the polygynous bat
365 *Phyllostomus hastatus*. *Behav. Ecol. Sociobiol.* **8**, 11–34. (10.1007/bf00302840).
- 366 [27] Jones KE, Bielby J, Cardillo M, Fritz SA, O’Dell J, Orme CDL, Safi K, Sechrest W, Boakes EH,
367 Carbone C, et al. 2009. PanTHERIA: a species-level database of life history, ecology, and geography
368 of extant and recently extinct mammals. *Ecology* **90**, 2648–2648. (10.1890/08-1494.1).
- 369 [28] Kerth G 2008. Causes and consequences of sociality in bats. *Bioscience* **58**, 737–746. (10.1641/B580810).
- 370 [29] Calisher C, Childs J, Field H, Holmes K, and Schountz T 2006. Bats: important reservoir hosts of
371 emerging viruses. *Clin. Microbiol. Rev.* **19**, 531–545. (10.1128/CMR.00017-06).
- 372 [30] Li W, Shi Z, Yu M, Ren W, Smith C, Epstein JH, Wang H, Crameri G, Hu Z, Zhang H, et al.
373 2005. Bats are natural reservoirs of SARS-like coronaviruses. *Science* **310**, 676–679. (10.1126/sci-
374 ence.1118391).
- 375 [31] Munywoki PK, Koech DC, Agoti CN, Kibirige N, Kipkoech J, Cane PA, Medley G, and Nokes DJ
376 2015. Influence of age, severity of infection, and co-infection on the duration of respiratory syncytial
377 virus (RSV) shedding. *Epidemiol. Infect.* **143**, 804–812. (10.1017/S0950268814001393).
- 378 [32] Halpin K, Hyatt AD, Fogarty R, Middleton D, Bingham J, Epstein JH, Rahman SA, Hughes T,
379 Smith C, Field HE, et al. 2011. Pteropid bats are confirmed as the reservoir hosts of henipaviruses:
380 a comprehensive experimental study of virus transmission. *Am. J. Trop. Med. Hyg.* **85**, 946–951.
381 (10.4269/ajtmh.2011.10-0567).
- 382 [33] De Thoisy B, Bourhy H, Delaval M, Pontier D, Dacheux L, Darcissac E, Donato D, Guidez A,
383 Larrous F, Lavenir R, et al. 2016. Bioecological drivers of rabies virus circulation in a neotropical bat
384 community. *PLoS Negl. Trop. Dis.* **10**. Ed. by Kasper M, e0004378. (10.1371/journal.pntd.0004378).
- 385 [34] Burns LE and Broders HG 2014. Correlates of dispersal extent predict the degree of population
386 genetic structuring in bats. *Conserv. Genet.* **15**, 1371–1379.
- 387 [35] R Development Core Team 2010. *R: A language and environment for statistical computing*. R
388 Foundation For Statistical Computing. Vienna, Austria. (<http://www.R-Project.org>).
- 389 [36] Lucas TCD 2015. MetapopEpi: Functions to run multipathogen, metapopulation epidemiological
390 simulations. R package version 0.0.1. (10.5281/zenodo.48942). (<https://github.com/timcdlucas/metapopepi>).
- 391 [37] Kermack WO and McKendrick AG 1932. Contributions to the Mathematical Theory of Epidemics.
392 II. The Problem of Endemicity. *Proc. R. Soc. A* **138**, 55–83. ISSN: 0950-1207. (10.1098/rspa.1932.0171).

- 393 [38] Plowright RK, Eby P, Hudson PJ, Smith IL, Westcott D, Bryden WL, Middleton D, Reid PA,
394 McFarlane RA, Martin G, et al. 2015. Ecological dynamics of emerging bat virus spillover. *Proc. R.*
395 *Soc. B* **282**, 20142124. (10.1098/rspb.2014.2124).
- 396 [39] Amengual B, Bourhy H, López-Roig M, and Serra-Cobo J 2007. Temporal dynamics of European
397 bat Lyssavirus type 1 and survival of *Myotis myotis* bats in natural colonies. *PLoS One* **2**, e566.
398 (10.1371/journal.pone.0000566).
- 399 [40] Peel A, Baker K, Crameri G, Barr J, Hayman D, Wright E, Broder C, Fernández-Loras A, Fooks A,
400 Wang L, et al. 2012. Henipavirus neutralising antibodies in an isolated island population of African
401 fruit bats. *PLoS One* **7**, e30346. (10.1371/journal.pone.0030346).
- 402 [41] Sohayati A, Hassan L, Sharifah S, Lazarus K, Zaini C, Epstein JH, Naim NS, Field HE, Arshad
403 SS, Aziz JA, et al. 2011. Evidence for Nipah virus recrudescence and serological patterns of captive
404 *Pteropus vampyrus*. *Epidemiology and Infection* **139**, 1570–1579. (10.1017/S0950268811000550).
- 405 [42] Rahman S, Hassan S, Olival K, Mohamed M, Chang L, Hassan L, Saad N, Shohaimi S, Mamat Z,
406 Naim M, et al. 2010. Characterization of Nipah virus from naturally infected *Pteropus vampyrus*
407 bats, Malaysia. *Emerg. Infect. Dis.* **16**, 1990.
- 408 [43] Chen SF, Jones G, and Rossiter S 2008. Sex-biased gene flow and colonization in the Formosan
409 lesser horseshoe bat: inference from nuclear and mitochondrial markers. *J. Zool.* **274**, 207–215.
- 410 [44] O'Donnell CF, Richter S, Dool S, Monks JM, and Kerth G 2015. Genetic diversity is maintained
411 in the endangered New Zealand long-tailed bat (*Chalinolobus tuberculatus*) despite a closed social
412 structure and regular population crashes. *Conserv. Genet.* 1–12. (10.1007/s10592-015-0746-9).
- 413 [45] Vonhof MJ, Russell AL, and Miller-Butterworth CM 2015. Range-wide genetic analysis of little
414 brown bat (*Myotis lucifugus*) populations: estimating the risk of spread of white-nose syndrome.
415 *PLoS One* **10**. (10.1371/journal.pone.0128713).
- 416 [46] Peel AJ, Sargan DR, Baker KS, Hayman DT, Barr JA, Crameri G, Suu-Ire R, Broder CC, Lembo
417 T, Wang LF, et al. 2013. Continent-wide panmixia of an African fruit bat facilitates transmission
418 of potentially zoonotic viruses. *Nat. Commun.* **4**. (10.1038/ncomms3770).
- 419 [47] Petit E and Mayer F 1999. Male dispersal in the noctule bat (*Nyctalus noctula*): where are the
420 limits? *Proc. R. Soc. B* **266**, 1717–1722. (10.1098/rspb.1999.0837).
- 421 [48] Brierley L, Vonhof M, Olival K, Daszak P, and Jones K 2016. Quantifying global drivers of zoonotic
422 bat viruses: a process-based perspective. *Am. Nat.* **187**, E53–E64. (10.1086/684391).
- 423 [49] Lindenfors P, Nunn CL, Jones KE, Cunningham AA, Sechrest W, and Gittleman JL 2007. Parasite
424 species richness in carnivores: effects of host body mass, latitude, geographical range and population
425 density. *Global. Ecol. Biogeogr.* **16**, 496–509. (10.1111/j.1466-8238.2006.00301.x).
- 426 [50] Vickery WL and Poulin R 1998. Parasite extinction and colonisation and the evolution of parasite
427 communities: a simulation study. *Int. J. Parasitol.* **28**, 727–737. (10.1016/S0020-7519(98)00036-8).
- 428 [51] Rivera-Ortíz F, Aguilar R, Arizmendi M, Quesada M, and Oyama K 2015. Habitat fragmentation
429 and genetic variability of tetrapod populations. *Anim. Conserv.* **18**, 249–258. (10.1111/acv.12165).

-
- 430 [52] Fontúrbel FE, Candia AB, Salazar DA, Malebrán J, González-Browne C, and Botto-Mahan C 2014.
431 How forest marsupials are affected by habitat degradation and fragmentation? A meta-analysis.
432 *Naturwissenschaften* **101**, 599–602. (10.1007/s00114-014-1193-z).
- 433 [53] Lehmann J, Korstjens AH, and Dunbar RI 2010. Apes in a changing world — the effects of
434 global warming on the behaviour and distribution of African apes. *J. Biogeogr.* **37**, 2217–2231.
435 (10.1111/j.1365-2699.2010.02373.x).
- 436 [54] Atwood TC 2006. The influence of habitat patch attributes on coyote group size and interaction
437 in a fragmented landscape. *Can. J. Zool.* **84**, 80–87. (10.1139/z05-180).
- 438 [55] Walters CL, Collen A, Lucas T, Mroz K, Sayer CA, and Jones KE 2013. “Challenges of using
439 bioacoustics to globally monitor bats”. In: *Bat Evolution, Ecology, and Conservation*. Ed. by Adams
440 RA and Pedersen SC. Springer, 479–499. (10.1007/978-1-4614-7397-8_23).
- 441 [56] Measey GJ, Stevenson BC, Scott T, Altwegg R, and Borchers DL 2016. Counting chirps: acoustic
442 monitoring of cryptic frogs. *Journal of Applied Ecology*. ISSN: 1365-2664. (10.1111/1365-2664.12810).
443 (<http://dx.doi.org/10.1111/1365-2664.12810>).
- 444 [57] Lucas TCD, Moorcroft EA, Freeman R, Rowcliffe JM, and Jones KE 2015. A generalised random
445 encounter model for estimating animal density with remote sensor data. *Methods Ecol. Evol.* **6**, 500–
446 509. (10.1111/2041-210X.12346).
- 447 [58] Altizer S, Nunn CL, and Lindenfors P 2007. Do threatened hosts have fewer parasites? A compar-
448 ative study in primates. *J. Anim. Ecol.* **76**, 304–314. (10.1111/j.1365-2656.2007.01214.x).